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Short communication

Bound to supine sleep: Parkinson's disease and the impact of nocturnal immobility

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ABSTRACT

Background: Impaired nocturnal mobility is a well-known problem in Parkinson's disease (PD), and clinical experience suggests a predominance of supine body position during sleep. However, this assumption – and potential consequences – still awaits objective validation by a polysomnography-based and adequately controlled study.
Methods: Clinical and polysomnographical analysis of 80 consecutive PD patients and 80 control subjects carefully matched for age, sex, body mass index and apnea–hypopnea index.
Results: PD patients slept twice as much in supine position than control subjects (62.2 ± 32.9% vs. 34.2 ± 28.5%, $p < 0.001$). In PD, but not in control subjects, more supine sleep correlated with fewer changes in body position (rho = −0.434, $p < 0.001$). Longer PD disease duration was an independent predictor of more supine sleep in multiple linear regression analysis ($\beta = 0.389$, $p < 0.001$); conversely, more supine sleep was associated with higher apnea–hypopnea index and daytime sleepiness.
Conclusions: We confirmed that supine sleep is common in PD, and increases with longer disease duration. Our findings indicate that supine sleep may contribute to the overall disease burden by deteriorating sleep-disordered breathing and daytime vigilance.

1. Introduction

A myriad of motor and non-motor symptoms add to the overall clinical burden in Parkinson's disease (PD), and sleep–wake disturbances are among the most frequent and significant contributors [1]. Patients often complain about difficulty turning in bed and claim to sleep mostly in supine position. However, whether PD patients do sleep more in supine position is unclear, because researchers have rarely analyzed the distribution of sleep positions in PD. In contrast, nocturnal immobility is recognized as a common disease feature, and an item pertaining to this symptom has been included in several clinical scales, including the Parkinson's Disease Quality of Life Questionnaire and the old and new versions of the Unified Parkinson's Disease Rating Scale [2].

Few studies reported adverse effects of nocturnal immobility on sleep quality in PD, whereas a recent polysomnography-based study failed to detect differences in the distribution of sleep positions in PD patients with and without subjectively impaired bed mobility [3,4]. Overall, most of these studies were based only on subjective complaints and lacked appropriate control groups.

Thus, in the present study we aimed 1) at objectively comparing the distribution of distinct sleep positions and the frequency of body position changes between PD patients and carefully matched control subjects, and 2) at identifying clinical correlates and predictors of supine sleep position in PD.

2. Patients and methods

2.1. Subjects, clinical assessment and matching procedure

This was a retrospective chart review. In a first step, we selected 119 consecutive PD patients who received whole-night video-polysomnography (PSG) as an integral diagnostic procedure between 2004 and 2009. In order to compare their data on sleep positions and nocturnal mobility to an adequate control group, we considered 248 consecutive non-neurological patients from our data base, in which PSG was performed during the same time for suspected

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sleep disorders. We used propensity score matching as a rater-independent and unbiased method to generate two groups accurately matched for age, sex, body mass index (BMI, body weight divided by the square of individual's height) and apnea–hypopnea index (AHI, number of apnea and hypopnea events per hour of sleep) [5]. By this means, we eventually included 80 PD patients and 80 matched control subjects.

Diagnostic procedure, clinical assessment and calculation of levodopa dose equivalents (LDE) are identical to previous studies [5]. We ascertained sleepiness using the Epworth Sleepiness Scale (ESS), with scores of $\geq$10 indicating excessive daytime sleepiness (EDS). The study was approved by the local Ethics Committee.

2.2. Polysomnography assessment

All patients had overnight PSG recordings, using a multichannel recording system (Embla, RemLogic™). Scoring of sleep stages and respiratory events was performed visually using standardized criteria [5]. Apnea was defined by a cessation of oro-nasal airflow longer than 10 s, and hypopnea by a reduction of oro-nasal airflow by at least 50% lasting more than 10 s and accompanied by an arousal or SaO_2 reduction of $\geq$3%. A body position sensor was attached by an elastic velcro strap at the level of the lower sternum and recognized distinct body positions by virtue of different voltage outputs. This allowed for automatic detection of the following four body positions: supine, right, left, and prone. In theory, the sensor would also register upright body position, but our subjects did not sleep in this position. We ensured adequate monitoring by comparing patient video and body position recording. As an objective measure of nocturnal immobility we used the number of changes between distinct body positions per hour.

2.3. Statistical analysis

Statistical analyses were performed using SPSS (version 21). Group data were described by means and standard deviations. For normally distributed data, we used Student's *t*-test and for non-parametric data the Mann–Whitney *U*-test. Chi-square test was used for nominal data. We calculated Pearson's r for correlation analysis of normally and Spearman-rho for non-normally distributed data. Stepwise multiple linear regression analysis was done including age, sex, body mass index, disease duration, Hoehn and Yahr stage, disease type, use of dopamine agonist, use of long acting dopaminergic medication, and total LDE as independent variables. Significance was accepted at $p < 0.05$.

3. Results

3.1. Clinical and polysomnographic findings

Demographic, clinical and polysomnographic findings are summerized in Table 1. PD patients slept roughly twice as much in supine position (62.2 ± 32.9% vs. 34.2 ± 28.5%, $p < 0.001$), and had fewer body position changes than controls (4.2 ± 7.1/h vs. 4.5 ± 7.2/h, $p = 0.04$). Lying most in supine position during sleep ($\geq$90%) was more common among PD patients than controls (32% vs. 6%, $p < 0.001$) (Fig. 1A). More supine sleep correlated with fewer body position changes in PD (rho = −0.434, $p < 0.001$) but not in controls (rho = −0.200, $p = 0.08$).

3.2. Correlates of supine sleep position

PD patients with EDS spent more time in supine position than those without EDS (51.0 ± 35.2% vs. 75.4 ± 25.0%, $p = 0.001$), whereas controls did not differ in this respect (30.9 ± 31.1% vs. 38.8 ± 23.1%, $p = 0.07$) (Fig. 1B). In PD, but not in controls, multiple linear regression analysis revealed that more supine sleep was independently associated with higher AHI ($\beta = 0.260$, $p = 0.015$ and age as second significant associate: $\beta = 0.349$, $p = 0.001$). Periodic limb movements in sleep did not correlate with supine position in either group. Likewise, supine sleep position did not correlate with any sleep stage, sleep efficiency or arousal index, and group comparison did not reveal any difference in supine sleep between PD patients with various disease type (akinetic–rigid, tremor-dominant, equal). Finally, the use of dopamine agonists had no impact on total amount of supine position, and correlation analysis between LDE of dopamine agonists and of long-acting dopaminergic drugs at bedtime with supine position did not demonstrate any significant associations.

3.3. Predictors of supine sleep in PD

Linear regression analysis identified longer disease duration to be independently associated with more supine sleep in PD ($\beta = 0.389$, $p < 0.001$) (Fig. 1C).

4. Discussion

Compared to carefully matched control subjects, PD patients slept almost twice as much in supine position and less often changed nocturnal body posture. In addition, we identified longer PD duration as independent predictor of more supine sleep. These results have been expected, and may be regarded as inevitable consequences of nocturnal immobility and axial rigidity. However, more than confirming a commonplace, our study suggests relevant clinical implications of this predominance for supine sleep position, as indicated by the association with increased severity of sleep-disordered breathing and excessive daytime sleepiness.

Our knowledge about the distribution of sleep positions in general is limited, but the clinical impact of certain sleep positions has been acutely revealed by the discovery that prone sleep position is associated with a more than threefold increased risk of sudden infant death syndrome. PSG studies demonstrated that up to two thirds of patients with acute stroke spend the first nights nearly entirely in supine position, while the magnitude of supine sleep is likely to diminish following recovery [6]. In stroke patients, supine position contributes to sleep disordered breathing (SDB) and impacts functional outcome and mortality. Application of continuous positive airway pressure (CPAP) or positional treatment is therefore increasingly advocated [7].

In this line, our results and those of many other groups confirm that SDB is the most obvious problem caused by lying in supine position. Supine position increases the risk of upper airway collapses, causes more and longer apnoeic events, and necessitates a higher pressure from CPAP devices [8,9]. Cheyne–Stokes respiration, obstructive and central SDB all ameliorate after changing from supine to lateral sleep position. Thus, our study indicates that sleep-related respiration in PD is similarly susceptible to the adverse effects of supine position. Although the contribution of SDB to daytime sleepiness has been questioned in PD, a recent randomized placebo-controlled study in PD patients with SDB demonstrated a significant improvement of both nocturnal sleep consolidation and objective daytime sleepiness in those receiving CPAP treatment [10]. Thus, feasibility and efficacy of positional treatment – already a well established alternative to CPAP in patients with positional SDB [11] – need to be evaluated also in PD patients.

On the other hand, it is unlikely that increased SDB severity represents the sole explanation for the negative association between supine sleep and daytime sleepiness. Even healthy people with poor sleep quality spend more time in supine position than